

Review on Rosuvastatin

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Review Article

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ABSTRACT

Rosuvastatin utilised in combination with exercise, diet, and weight-loss to treat high steroid alcohol and connected conditions, and to prevent upset. Rosuvastatin is metabolized in the main by CYP2C9 and not extensively metabolized; or so ten is recovered as substance. It's excreted in excretion (90%) primarily and therefore the elimination half-life is or so nineteen h. Pharmacodynamics: Rosuvastatin may be a selective and competitive matter of reductase, the rate-limiting accelerator that converts 3-hydroxy-3-methylglutaryl coenzyme A to mevalonate, a precursor of sterol. *in vivo* studies in animals, and *in vitro* studies in polite animal and human cells have shown rosuvastatin to possess a high uptake into, and property for, action within the liver, the organ for sterol lowering. Rosuvastatin is employed in conjunction with a correct diet to assist lower "bad" sterol and fats (such as low-density lipoprotein, triglycerides) and lift "good" sterol (HDL) within the blood. It belongs to a gaggle of medication referred to as "statins." It works by reducing the number of sterol created by the liver. Lowering "bad" sterol and triglycerides and raising "good" sterol decreases the chance of cardiopathy and helps to forestall strokes and heart attacks.

INTRODUCTION

Cholesterol is a waxy, fat-like substance that is found in all cells of the body. Your body needs some cholesterol to make hormones, vitamin D, and substances that help you process nourishments. Your body makes all the cholesterol it needs ^[1]. Be that as it may, cholesterol likewise is found in a portion of the sustenance's you eat. Cholesterol goes through your circulatory system in little bundles called lipoproteins (lip-o-PRO-teenagers). These bundles are made of fat (lipid) within and proteins on the outside. Two sorts of lipoproteins convey cholesterol all through your body: Low-thickness lipoproteins (LDL) and high-thickness lipoproteins (HDL). Having sound levels of both sorts of lipoproteins is important. LDL cholesterol here and there is called "awful" cholesterol. A high LDL level prompts a development of cholesterol in your veins (Conduits are veins that convey blood from your heart to your body) ^[2,3]. HDL cholesterol some of the time is called "great" cholesterol. This is on account of it conveys cholesterol from different parts of your body back to your liver. Your liver expels the cholesterol from your body.

High blood cholesterol is a condition in which you have an excessive amount of cholesterol in your blood. Without anyone else, the condition more often than not has no signs or side effects. In this way, numerous individuals don't have the foggiest idea about that their cholesterol levels are excessively high ^[4-9]. People who have high blood cholesterol have a more noteworthy shot of getting coronary illness, additionally called coronary conduit infection (In this article, the expression "coronary illness" alludes to coronary heart disease). The higher the level of LDL cholesterol in your blood, the GREATER your chance is of getting coronary illness. The higher the level of HDL cholesterol in your blood, the LOWER your chance is of getting heart disease. Coronary heart disease is a condition in which plaque (plak) develops inside the coronary (heart) supply routes. Plaque is comprised of cholesterol, fat,

calcium, and different substances found in the blood [10-16]. At the point when plaque develops in the courses, the condition is called atherosclerosis (ATH-er-o-skler-O-sister).

Medication is one of the alternatives to treat high cholesterol. Some of the anti-cholesterol drugs are atorvastatin (Lipitor) [17], fluvastatin (Lescol, Lescol XL), lovastatin (Mevacor, Altoprev), pravastatin (Pravachol), rosuvastatin (Crestor), simvastatin (Zocor), and pitavastatin (Livalo). Rosuvastatin, marketed as Crestor, may be a member of the drug category of statins, utilized in combination with exercise, diet, and weight-loss to treat high cholesterol and connected conditions, and to stop upset [18-22]. The first use of rosuvastatin is for the treatment of dyslipidemia. Effects on cholesterol levels: The effects of rosuvastatin on LDL cholesterol square measure dose-related. Higher doses were a lot of efficacious in up the macromolecule profile of patients with hypercholesteremia than milligram-equivalent doses of statin and milligram-equivalent or higher doses of Zocor and statin drug. Meta-analysis showed that rosuvastatin is in a position to with modestly increase levels of cholesterol additionally, like different statins [19,23-27]. A 2014 Cochrane review determined there was smart proof for rosuvastatin lowering non-HDL levels linearly with dose. Alpha-lipoprotein will increase by seven-membered with no dose result noted. Rosuvastatin has structural similarities with most different artificial statins, e.g., statin, Baycol and pitavastatin, however in contrast to different statins rosuvastatin contains sulphur [2,5,28-33]. Crestor is truly rosuvastatin metal, within which metal replaces the element within the acid cluster. The main Mechanism of rosuvastatin is: Rosuvastatin may be a competitive substance of the accelerator reductase, having a mechanism of action like that of different statins. Its approximate elimination half-life is nineteen h and it's time to peak plasma concentration is reached in 3-5 h following oral administration [34]. Putative helpful effects of rosuvastatin medical care on chronic coronary failure is also negated by will increase in albuminoid turnover markers additionally as a discount in plasma molecule Q10 levels in patients with chronic coronary failure [24,29,32,35].

BIOAVAILABILITY

It is observed that the absolute bioavailability of rosuvastatin is ready 20% and C_{max} is reached in three to five h; control with food did not have an effect on the AUC in line with the actual sponsor submitted scientific examine and as in keeping with product label [36-43]. However, a subsequent clinical test has shown a marked discount in rosuvastatin exposure while administered with meals. Its miles 88 percent protein bound, particularly to albumin. Fraction absorbed of rosuvastatin is frequently misquoted inside the literature as about 0.5 (50%) because of a miscalculated hepatic extraction ratio inside the unique submission bundle in the end corrected by using the use of the FDA reviewer [44-50].

Rosuvastatin is metabolized primarily by CYP2C9 and not extensively metabolized; close to ten is recovered as matter. It's excreted in excreta (90%) primarily and therefore the elimination half-life is close to nineteen h [51-56]. Rosuvastatin may be a selective and competitive matter of 5-hydroxy-3-methylglutaryl-coenzyme A reductase, the rate-limiting accelerator that converts 3-hydroxy-3-methylglutaryl coenzyme A to mevalonate, a precursor of steroid alcohol [57-62]. *In vivo* studies in animals and *in vitro* studies in cultivated animal and human cells have shown rosuvastatin to possess a high uptake into, and property for, action within the liver, the organ for steroid alcohol lowering. Rosuvastatin exerts its lipid-modifying effects by increasing the amount of internal organ rarity compound protein (LDL) receptors on the cell surface, enhancing uptake and organic process of (LDL) low-density compound protein. LDL β -lipoprotein lipoprotein and inhibiting the internal organ synthesis of terribly rarity lipoprotein, thereby reducing the full very low density lipoprotein (VLDL) and LDL particles [53,59,63-70]. Studies have shown that 5-hydroxy-3-methylglutaryl-coenzyme A reductase inhibitors decrease non-high density compound protein (non-HDL) id est, all current steroid alcohol not in lipoprotein, and apolipoprotein B (Apo B) or cut back the Apo B/Apo A-1 quantitative relation.

Pharmacokinetics

As observed the height of rosuvastatin plasma concentrations area unit reached 3-5 h once oral administration. Each peak concentration (C_{max}) and space underneath the plasma concentration-time curve (AUC) increase in direct proportion to rosuvastatin dose [71-75]. Absolute bioavailability of rosuvastatin is just about 2 hundredth. Administration of rosuvastatin with food didn't have an effect on rosuvastatin United Self-Defense Force of Colombia. Rosuvastatin United Self-Defense Force of Colombia doesn't take issue once morning or evening drug administration. Rosuvastatin's mean volume of distribution at steady-state is regarding 134 L. *Rosuvastatin* is half a milesure to plasma proteins, principally simple protein. This binding is reversible and freelance of plasma

concentrations. Rosuvastatin isn't extensively metabolized; regarding ten of a radiolabeled dose is recovered as substance [76-80]. The foremost substance is N-desmethyl rosuvastatin, that is created primarily by hemoprotein P450 2C9, and *in vitro* studies have incontestable that N-desmethyl rosuvastatin has just about 1/6-1/2 the HMG-CoA reductase restrictive activity of rosuvastatin. Overall, >90% of active plasma HMG-CoA reductase restrictive activity is accounted for by rosuvastatin [81-83]. Rosuvastatin and its metabolites are primarily excreted within the faeces (90%) once oral administration. It is also observed that the elimination half-life ($t_{1/2}$) is regarding nineteen hrs. Pharmacokinetic studies demonstrate an approximate 2-fold increase in median exposure (AUC and C_{max}) in Asian subjects (having either Vietnamese, Filipino, Chinese, Japanese, Korean or Asian-Indian origin) compared with Caucasians. A population pharmacokinetic analysis showed no clinically relevant variations in pharmacological medicine among Caucasians, Hispanic and Black, or Afro-Caribbean teams [84-89].

USES

Rosuvastatin is employed in conjunction with a correct diet to assist lower "bad" cholesterol and fats (such as β -lipoprotein, triglycerides) and lift "good" cholesterol (HDL) within the blood. It belongs to a gaggle of medicine referred to as "statins." It works by reducing the quantity of cholesterol created by the liver. Lowering "bad" cholesterol and triglycerides and raising "good" cholesterol decreases the danger of cardiovascular disease and helps to stop strokes and heart attacks [89-91]. In addition to ingestion a correct diet (such as an occasional cholesterol/low-fat diet), alternative style changes which will facilitate this medication work higher embrace elbow grease, losing weight if overweight, and stopping smoking.

HOW TO USE ROSUVASTATIN?

Take this medication orally with or while not food as directed by your doctor, typically once daily. The dose is predicated on your medical condition, response to treatment, age, race, and alternative medications you'll be taking. Take care to inform your doctor and health professional regarding all the merchandise you utilize (including pharmaceuticals, non-prescription medicine, and flavouring products). If you're of Asian descent, your doctor might direct you to start out with a lower dose as a result of you'll be additional sensitive to its effects [7,9,32,46,92-95]. Antacids containing aluminium or Mg will cut back the absorption of this drug. Therefore, if taking this kind of antacid, take it a minimum of a pair of hours when this medication. Take this medication often so as to urge the foremost get pleasure from it bear in mind to require it at identical time every day. It's vital to continue taking this medication though you are feeling well. The general public with high cholesterol or triglycerides doesn't feel sick. It's vital to still follow your doctor's recommendation regarding diet and exercise. It is going to take up to four weeks before you get the complete advantage of this drug.

WHAT CONDITIONS DOES ROSUVASTATIN TREAT?

A very tiny variety of individuals taking rosuvastatin could have gentle memory issues or confusion. If these rare effects occur, refer to your doctor. Rarely, statins could cause or worsen polygenic disease. This drug could seldom cause muscle issues (which will seldom result in terribly serious conditions known as rhabdomyolysis and reaction myopathy) [96]. Tell your doctor at once if you develop any of those symptoms throughout treatment and if these symptoms persist once your doctor stops this drug: muscle pain/tenderness/weakness (especially with fever or uncommon tiredness), signs of excretory organ issues (such as modification within the quantity of urine).

SIDE EFFECTS

This medication could seldom cause liver issues. If you notice any of the subsequent rare however serious aspect effects, yellowing eyes/skin, dark urine, severe stomach/abdominal pain, persistent nausea/vomiting. A very serious hypersensitive reaction to the present drug is rare. However, get medical facilitate at once if any symptoms of a significant hypersensitive reaction, including: rash, itching/swelling (especially of the face/tongue/throat), dizziness, bother respiration is noticeable [11,13,72,97].

INTERACTIONS

Drug interactions could amend however your medications work or increase your risk for serious aspect effects. An inventory of the entire product used ought to be maintained (including prescription/non-

prescription medicine and flavouring products) and will be shared to the doctor and health professional. Must not be begin, stop, or amendment the indefinite quantity of any medicines while not your doctor's approval [98- 99]. Some product that will move with this drug include: "Blood thinners" (such as warfarin), Lopid. Other medications will have an effect on the removal of rosuvastatin from your body, which can have an effect on however rosuvastatin works. Examples embrace ledipasvir, among others.it is suggested to not take any red yeast rice product whereas you're taking rosuvastatin as a result of some red yeast rice product might also contain a medicament known as lipid-lowering medication [6,47,88,91]. Taking rosuvastatin and red yeast rice product along will increase your risk of great muscle and liver issues.

CONCLUSION

Rosuvastatin used together with exercise, diet, and weight-loss to treat high steroid and connected conditions, and to stop upset. Rosuvastatin is utilized in conjunction with an accurate diet to help lower "bad" sterol and fats (such as LDL, triglycerides) and elevate "good" sterol (HDL) among the blood. It belongs to a gaggle of medication stated as "statins." It works by reducing the number of sterol created by the liver [6,25,55]. Lowering "bad" sterol and triglycerides and raising "good" sterol decreases the danger of upset and helps to prevent strokes and heart attacks. Additionally to consumption an accurate diet (such as Associate in nursing occasional cholesterol/low-fat diet), various vogue changes which can facilitate this medication work higher embrace labor, losing weight if overweight, and stopping smoking. This medication may rarely cause liver problems. If you notice any of the following rare but serious side effects, yellowing eyes/skin dark urine, severe stomach/abdominal pain, persistent nausea /vomiting. An awfully serious allergic reaction to this drug is rare [62,84,98-100]. However, get medical facilitate quickly if any symptoms of a major allergic reaction, including: rash, itching/swelling (especially of the face/tongue/throat), dizziness, hassle respiration is noticed.

REFERENCES

1. Ohvo-Rekilä H, et al. "Cholesterol interactions with phospholipids in membranes". *Prog Lipid Res.* 2002;41:66-97.
2. Olson RE. Discovery of the lipoproteins, their role in fat transport and their significance as risk factors. *J Nutr.* 1998;128:439S-443S.
3. William C. Lipid analysis: isolation, separation, identification, and structural analysis of lipids. Ayr, Scotland: Oily Press;2003.
4. Lewis GF and Rader DJ. New insights into the regulation of HDL metabolism and reverse cholesterol transport. *Circ Res.* 2005;96:1221-1232.
5. Gordon DJ, et al. High-density lipoprotein cholesterol and cardiovascular disease. Four prospective American studies. *Circulation.* 2005;79:8-15.
6. Wang TY, et al. Hypercholesterolemia paradox in relation to mortality in acute coronary syndrome. *Clin Cardiol.* 2009;32:E22-E28.
7. Warnick GR, et al. Estimating low-density lipoprotein cholesterol by the Friedewald equation is adequate for classifying patients on the basis of nationally recommended cutpoints. *Clin Chem.* 1990;36:15-19.
8. Aggarwal RK and Showkathali R. Rosuvastatin calcium in acute coronary syndromes. *Expert Opinion on Pharmacotherapy.* 2013;14:1215-1227.
9. Jones PH, et al. Comparison of the efficacy and safety of rosuvastatin versus atorvastatin, simvastatin, and pravastatin across doses (STELLAR Trial). *Am J Cardiol.* 2003;92:152-160.
10. McTaggart F. Effects of statins on high-density lipoproteins: a potential contribution to cardiovascular benefit. *Cardiovasc Drugs Ther.* 2008;22:321-338.
11. Adams SP, et al. Lipid-lowering efficacy of rosuvastatin. *Cochrane Database Syst Rev.* 2014;11:CD010254.
12. Nissen SE, et al. Effect of very high-intensity statin therapy on regression of coronary atherosclerosis: the ASTEROID trial. *JAMA.* 2006;295:1556-1565.
13. Li Y, et al. Pharmacokinetic Properties of Rosuvastatin After Single-Dose, Oral Administration in Chinese Volunteers: A Randomized, Open-Label, Three-Way Crossover Study. *Clinical Therapeutics.* 2007;29:2194-2203.
14. Ebba B, et al. Enterohepatic Disposition of Rosuvastatin in Pigs and the Impact of Concomitant Dosing with Cyclosporine and Gemfibrozil. *Drug Metabolism and Disposition.* 2009;37:2349-2358.
15. Ridker PM, et al. Rosuvastatin to Prevent Vascular Events in Men and Women with Elevated C-Reactive Protein. *N Engl J Med.* 2008;359:2195-2207.

16. Fellstrom BC, et al. Rosuvastatin and Cardiovascular Events in Patients Undergoing Hemodialysis. *N Engl J Med*. 2009;360:1395-1407.
17. Sanai T, et al. Benefits and Adverse Effects of Statins, Atorvastatin Calcium Hydrate, Pitavastatin Calcium, and Pravastatin Sodium, for Dyslipidemia in Patients on Hemodialysis. *J Metabolic Syndr*. 2016;5:202.
18. Mohamed AA, et al. Statins Added to Chronic Hepatitis C Treatment: Is it Beneficial? *J Hepatol Gastroint Dis*. 2016;2:117.
19. Weber SU, et al. Statins Downregulate the Constitutive Expression of HLA-DR and Reduce Intracellular CD74 in the Monocyte Cell Line Mono Mac 6. *Biochem Pharmacol*. 2016;5:200.
20. Diaconu CC. Statins in Patients with Chronic Kidney Disease: To Give or Not To Give. *Pharm Anal Acta*. 2015;6:448.
21. Bagchi S, et al. Underutilization of Statins for Prevention of Cardiovascular Disease among Primarily African-American HIV-Infected Patients. *J AIDS Clin Res*. 2015;6:499.
22. Shimoyama S. Statins' Cardiovascular Benefits Outweigh their Diabetogenicity: A Direct Comparison between Number Needed to Treat and Number Needed to Harm. *Adv Pharmacoepidemiol Drug Saf*. 2015; 4:185.
23. Dolkart O, et al. The Effects of Lipophilic and Hydrophilic Statins on Bone Tissue Mineralization in Saos2 Human Bone Cell Line–In vitro Comparative Study. *Pharm Anal Acta*. 2015;6:363.
24. Guidone D, et al. Sodium Fusidate Inhibits rCYP3A4 in vitro–A Possible Mechanism Defining the Interaction with Statins. *Clin Exp Pharmacol*. 2015;5:174.
25. Gadzhanova S and Roughead E. Co-prescribing of Warfarin with Statins and Proton Pump Inhibitors in Elderly Australians. *Adv Pharmacoepidemiol Drug Saf*. 2014;3:161.
26. Moore TL, et al. Polymer-Coated Hydroxyapatite Nanoparticles for the Delivery of Statins. *J Nanomed Nanotechnol*. 2014;5:237.
27. Heeba G, et al. Nanomedical Approach to Monitor the Central Role of NO/ONOO- Imbalance in Ischemic Stroke Brain Damage – The Effects of Statins and Heme Oxygenase-1. *J Nanomed Nanotechnol*. 2014;5:215.
28. Sahebzamani FM, et al. Examination of the FDA Warning for Statins and Cognitive Dysfunction. *J Pharmacovigilance*. 2014;2:141.
29. Kumar GP. The Potential of Statins for Buccal Delivery. *J Mol Pharm Org Process Res*. 2014;2:111.
30. Figg G, et al. Statins as a Primary Prevention: Which One is Most Effective? A Systematic Review and Meta-Analysis. *J Cardiovasc Dis Diagn*. 2013;1:109.
31. Patel A and Pisklakov SV. Statins as Potentially Neuroprotective Agents: A Review. *J Anesth Clin Res*. 2012;3:251.
32. Kelesidis T. Statins as Antiviral and Anti-inflammatory Therapy in HIV Infection. *Virology & Mycology*. 2012;1:e102.
33. Marchi RC. Statins Therapy: Effects on Plasma Fibrinogen Levels and Fibrinolysis. *J Nutr Disorders Ther*. 2012;S6:001.
34. Feher A, et al. Generic Statins in Cardiovascular Medicine. *J Bioequiv Availab*. 2011;S2.
35. Mehra S, Desai T (2011) Statins in Chronic Kidney Disease- Are Statins Really Renoprotective. *J Nephrol Therapeutic*. 1:103.
36. Della Bona R, De Caterina AR, Leo M, Biasillo G, Basile E (2011) Statins Reduce Incidence of Early Perioperative Complications and Length of in-Hospital Stay after Coronary Artery Bypass Graft Surgery. *J Clinic Experiment Cardiol* 2:137.
37. Tabassum A, et al. Synthetic Characterization of Complexes of Rosuvastatin and Some ACE Inhibitors: Pharmacological Evaluation. *Pharm Anal Acta*. 2016;7:488.
38. Niddam-Hildesheim V and Sterimbaum G. Teva Pharmaceutical Industries Ltd. Process for preparation of rosuvastatin calcium. 2008.
39. López-Canales JS, et al. The methyl ester of rosuvastatin elicited an endothelium-independent and 3-hydroxy-3-methylglutaryl coenzyme A reductase-independent relaxant effect in rat aorta. *Braz J Med Biol Res*. 2011;44:438-444.
40. Aronhime J and Hildesheim VN. Crystalline ammonium salts of rosuvastatin. Teva Pharmaceutical Industries Ltd, assignee. 2005.
41. Mak WY, et al. Pharmacokinetic Comparison and Bioequivalence Study of Two Rosuvastatin 20 mg Formulations in Healthy Volunteers. *J Bioequiv Availab*. 2006;8:95-98.
42. Gibson CM, et al. Effect of Intensive Statin Therapy on Clinical Outcomes Among Patients Undergoing Percutaneous Coronary Intervention for Acute Coronary Syndrome. *J Am Coll Cardiol*. 2009;54:2290-2295.

43. Vargas M, et al. Bioequivalence Study of Two Formulations Containing Rosuvastatin 40 Mg Tablets in Healthy Colombians. *J Bioequiv Availab*. 2015;7:229-232.
44. Ihoriya C, et al. Nuclear Factor Erythroid 2-Related Factor 2 is Activated by Rosuvastatin via p21cip1 Upregulation in Endothelial Cells. *Biochem Pharmacol*. 2014;4:157.
45. Mukthinuthalapati MA, et al. Simultaneous Determination of Rosuvastatin and Ezetimibe in pharmaceutical formulations by Stability Indicating Liquid Chromatographic Method. *J Bioequiv Availab*. 2014;6:174-180.
46. Naydenov SN, et al. Lipid-Lowering Potency and Tolerability of Generic Rosuvastatin in Bulgarian Patients with High and Very High Risk. *J Cardiovasc Dis Diagn*. 2014;2:162.
47. Rocha VN, et al. Beneficial Effects of Rosuvastatin in Heart of C57Bl/6 Mice with DietInduced Metabolic Syndrome - A Preliminary Study. *Endocrinol Metab Syndr*. 2014;3:121.
48. Mark L, et al. The Effect of Switching to the High-Efficient Rosuvastatin on the Success of Lipid Lowering Therapy in High Risk Patients. The CORVUS (Controlled Targets for High Vascular Risk Patients Using Effective Statins) Study. *Pharm Anal Acta*. 2013;4:267.
49. Sultana N, et al. An Ultra-Sensitive LC Method for Simultaneous Determination of Rosuvastatin, Alprazolam and Diclofenac Sodium in API, Pharmaceutical Formulations and Human Serum by Programming the Detector. *J Anal Bioanal Techniques*. 2012;3:154.
50. Khan SP, et al. Variation of Carotid Intima - Media Thickness in Hypercholesterolemia Patients on Atorvastatin and Rosuvastatin Therapy. *J Clin Exp Cardiol*. 2012;3:191.
51. Brugts JJ, et al. (2009). The benefits of statins in people without established cardiovascular disease but with cardiovascular risk factors: meta-analysis of randomized controlled trials. *BMJ*. 2009;338:b2376.
52. Nicholls SJ. Rosuvastatin and progression of atherosclerosis. *Expert Rev Cardiovasc Ther*. 2008;6:925-33.
53. Wanyama FM, et al. Evaluation of High Density Lipoprotein Cholesterol as a Predictor of Diabetic Nephropathy in Type 1 Diabetic Patients. *Clin Med Biochemistry*. 2016;2:117.
54. Kharb S, et al. Correlation of LDL Cholesterol with Maternal and Cord Blood Heme Oxygenase 1 in Preeclampsia. *J Preg Child Health*. 2016;3:225.
55. Edith NF, et al. Pleurotus florida Aqueous Extracts and Powder Influence Lipid Profile and Suppress Weight Gain in Rats Fed High Cholesterol Diet. *J Nutr Food Sci*. 2016;6:473.
56. Ozolua RI, et al. Extract of Garcinia kola Seed has Antitussive Effect and Attenuates Hypercholesterolemia in Rodents. *Med Aromat Plants*. 2016;5:232.
57. Naviglio D. Bad Cholesterol or "Bad" Science? *Med chem*. 2016;6:040.
58. Hari OS, et al. Awareness and Trends of Blood Cholesterol and Susceptibility to Develop Heart Disease. *Adv Genet Eng*. 2015;4:138.
59. Chowdhury S, et al. The Importance of Non High Density Lipoprotein Cholesterol in Dyslipidaemia Management. *J Diabetes Metab*. 2015;6:623.
60. Kader Sahib A, et al. Is Non - High-Density Lipoprotein Cholesterol (Non HDL-C) A Better Predictor Of Future Risk Of Cardiovascular Mortality? *Biochem Physiol*. 2015;4:e137.
61. Chen X, et al. Role of LDL Cholesterol and Endolysosomes in Amyloidogenesis and Alzheimer's Disease. *J Neurol Neurophysiol*. 2014;5:236.
62. Jansen EHJM, et al. Long Term Stability of Parameters of Lipid Metabolism in Frozen Human Serum: Triglycerides, Free Fatty Acids, Total-, HDL- and LDL-cholesterol, Apolipoprotein-A1 and B. *J Mol Biomark Diagn*. 2014;5:182.
63. Jhuma KA, et al. Effects of Atorvastatin and Niacin, Alone and in Combination, On Lowering Serum LDL-Cholesterol and Lipoprotein (a) in Hyperlipidemia Patients. *J Metabolic Syndr*. 2014;3:136.
64. Kamal SM. Aliskiren Protects against Hypercholesterolemia and Oxidative Stress on Isolated Aortae in Cholesterol-Fed Rats. *J Nanomed Nanotechol*. 2013;S5:007.
65. Ma LL, et al. Influence of Dietary Amino Acid Profile on Serum Lipids in Hypercholesterolemic Chinese Adults. *J Nutr Food Sci*. 2014;4:258.
66. Voloshyna I, et al. Advanced Glycation End Products Promote Pro-Atherogenic Changes in Cholesterol Transport: A Possible Mechanism for Cardiovascular Risk in Diabetes. *Intern Med*. 2014;S11:005.
67. Aye M, et al. Prevalence of Coronary Heart Disease among Non-Smokers with Type 2 Diabetes Mellitus and Metabolic Syndrome Defined By NCEPATP 111 (National Cholesterol Education Programme Adult Treatment Panel 111). *J Metabolic Syndr*. 2013;2:123.
68. Achudume AC, et al. Bioeffects of electromagnetic base station on glutathione reductase, lipid peroxidation and total cholesterol in different tissues of Wistar rats. *Biology and Medicine*. 2009;1:33-38.
69. Aronow WS. Treatment of Hypercholesterolemia. *J Clin Exp Cardiol*. 2013;S1:006.
70. Hadi NR, et al. Effect of Vildagliptin on Atherosclerosis Progression in High Cholesterol-Fed Male Rabbits. *J Clin Exp Cardiol*. 2013;4:249.
71. Shintani H. Determination of Cholesterol and its Oxides. *Pharm Anal Acta*. 2013;S1:003.

72. Khan SP, et al. Variation of Carotid Intima - Media Thickness in Hypercholesterolemia Patients on Atorvastatin and Rosuvastatin Therapy. *J Clin Exp Cardiol.* 2012;3:191.
73. Lamina S and Okoye GC. Therapeutic Role of Continuous Training Program on High Density Lipoprotein Cholesterol in Men with Hypertension: A Randomized Controlled Trial. *J Clinic Experiment Cardiol.* 2012;3:182.
74. Li R, et al. Gene Therapy Targeting LDL Cholesterol but not HDL Cholesterol Induces Regression of Advanced Atherosclerosis in a Mouse Model of Familial Hypercholesterolemia. *J Genet Syndr Gene Ther.* 2011;2:106.
75. Desplantie O, et al. Can Apolipoproteins apoB and apoB/apoA1 Ratio Predict Future Cardiovascular Risk Post Acute Coronary Syndrome? A Retrospective Cohort Study. *J Clin Exp Cardiol.* 2016;7:443.
76. Backes JM, et al. Evaluating the Effects of Prescription Fish Oil, Supplemental Fish Oil and a Krill Oil Blend on Serum Lipids/Lipoproteins and the Omega-3 Index: A Pilot Study. *J Glycomics Lipidomics.* 2014;4:121.
77. Vine DF, et al. Insulin and Testosterone are Associated with Elevated Intestinal Secretion of Lipids and Lipoproteins in a Rodent Model of the Metabolic and Polycystic Ovary Syndrome. *J Diabetes Metab.* 2014;5:391.
78. Spillmann F, et al. High density Lipoproteins Induce the Migration Capacity of Mesenchymal Stromal Cells. *J Stem Cell Res.* 2014;4:176.
79. Yu JY and Lyons TJ. Modified Lipoproteins in Diabetic Retinopathy: A Local Action in the Retina. *J Clin Exp Ophthalmol.* 2013;4:314.
80. Ebesunun MO, et al. Elevated plasma homocysteine in association with decreased vitamin B12, folate, serotonin, lipids and lipoproteins in depressed patients. *Afr J Psychiatry.* 2012;15:25-29.
81. Zhao Y, et al. Apolipoprotein E-Deficient Lipoproteins Induce Foam Cell Formation by Activation of PERKEIF-2a Signaling Cascade. *J Bioanal Biomed.* 2010;2:113-120.
82. Sun T, et al. Invasive Aortic Augmentation Index Could Predict the Adverse Events in Patients without Established Coronary Heart Disease. *Angiol.* 2016;4:173.
83. Roever L, et al. Exercise-Based Rehabilitation for Coronary Heart Disease: What does the Evidence Show? *J Cardiovasc Dis Diagn.* 2016;4:e111.
84. Wang L, et al. A Combination of Electro-Acupuncture and Aerobic Exercise Improves Cardiovascular Function in Patients with Coronary Heart Disease. *J Clin Exp Cardiol.* 2015;6:402.
85. Li G, et al. High Serum Concentration of Sulfatide is a Risk Factor for Restenosis in Patients with Coronary Heart Disease after Percutaneous Coronary Intervention. *Metabolomics.* 2015;5:137.
86. aduagu ATL, et al. Prevalence of Coronary Heart Diseases Risk Factors in Adults Population Living in Nigeria's Largest Urban City. *J Nutr Disorders Ther.* 2015;5:153.
87. Avila A, et al. A Randomized Controlled Study Comparing Home-Based Training with Telemonitoring Guidance Versus Center-Based Training in Patients with Coronary Heart Disease: Rationale and Design of the Tele-Rehabilitation in Coronary Heart Disease (TRiCH) Study. *J Clin Trials.* 2014;4:175.
88. Berkinbayev S, et al. Apolipoprotein Gene Polymorphisms (APOB, APOC111, APOE) in the Development of Coronary Heart Disease in Ethnic Groups of Kazakhstan. *J Genet Syndr Gene Ther.* 2014;5:216.
89. Zullo MD, et al. Factors Associated with Trying to Lose Weight in Women with Coronary Heart Disease: Do Factors Differ by Race/Ethnicity? *J Obes Weight Loss Ther.* 2013;3:196.
90. Virag J. New Twists on an Old Problem: Contemporary Experimental and Clinical Research of Coronary Heart Disease. *J Clin Exp Cardiol.* 2013;S6:007.
91. Aye M, et al. Prevalence of Coronary Heart Disease among Non-Smokers with Type 2 Diabetes Mellitus and Metabolic Syndrome Defined By NCEPATP 111 (National Cholesterol Education Programme Adult Treatment Panel 111). *J Metabolic Synd.* 2013;2:123.
92. de Jager CA. Why Don't We Know What to Eat in the 21st Century? A Focus on Coronary Heart Disease and Dementia Prevention. *Vitam Trace Elem.* 2013;2:e120.
93. Saldarriaga C. How to Reduce the Burden of Coronary Heart Disease in Women? *J Women's Health Care.* 2013;2:e111.
94. Suastika K, et al. Coronary Heart Disease in a Remote Area. *J Clin Exp Cardiol.* 2012;S6:002
95. Namekata T, Suzuki K, Ishizuka N, Nakata M, Shirai K (2012) Association of Cardio-Ankle Vascular Index with Cardiovascular Disease Risk Factors and Coronary Heart Disease among Japanese Urban Workers and their Families. *J Clinic Experiment Cardiol* S1:003.
96. Spencer-Hwang R, et al. Female Renal Transplant Recipients Potentially at Increased Risk of Fatal Coronary Heart Disease Associated with Ambient Air Pollutants. *J Clinic Experiment Cardiol.* 2011;S6:001.
97. Hanan H Hagar. Effects of rosuvastatin on the progression of diabetic nephropathy in streptozotocin-induced diabetic rats. 9th Diabetologists Conference. Dallas, Texas, USA. 2016.

98. Nidhi Garg. Rosuvastatin improves endothelial dysfunction in ankylosing spondylitis. 3rd International Conference and Exhibition on Pharmacovigilance & Clinical Trials. 2014 Hyderabad International Convention Centre, India
99. Trailokya AR. Evaluation and assessment of rosuvastatin 40mg treatment in high risk dyslipidemic patients (EARTH Study). 2nd International Summit on Clinical Pharmacy. 2014 Double Tree by Hilton Hotel San Francisco Airport, USA.
100. Boarelli P, et al. Simultaneous Study of Cholesterol and GM1 Ganglioside by Specific Probes: Lipid Distribution during Maturation, Capacitation and the Acrosome Reaction. *J Cytol Histol.* 2016;7:412.